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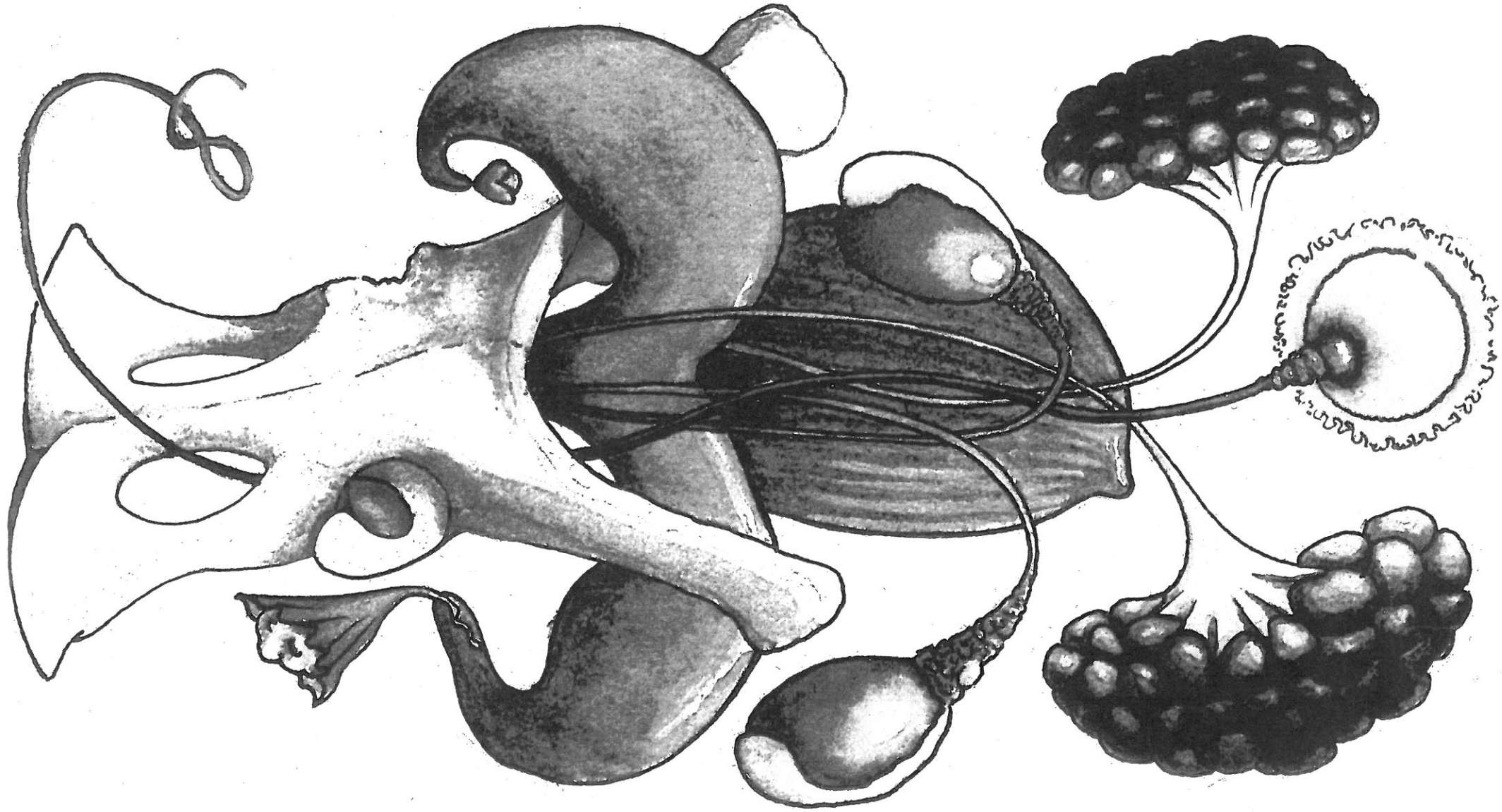

Spontaneous Endocarditis in Pigs - Pathology and Microbiology

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Introduction: The association between pathology and aetiology of endocarditis in pigs has only been elucidated in a few studies. Moreover, in most of these studies the endocardial lesions were obtained from experimentally infected animals. Therefore, only sporadic or textbook information is available on the pathology of porcine endocarditis. In the present study, the relation between aetiology and pathology of spontaneous porcine endocarditis is described.

Material and Methods: Hearts obtained during routine meat-inspection at 11 abattoirs were submitted for examination. Endocarditis lesions were recorded and material was sampled from the lesions for microbiological cultivation and pathology. Bacterial colonies were subsequently identified biochemically using standardized methods. Streptococci were further analyzed by means of partial 16S rRNA gene sequencing following PCR. For histology, tissues were fixed in 10% buffered formalin, trimmed, dehydrated, and embedded in paraffin wax. Sections of 3-4 µm were stained with haematoxylin and eosin (HE) and in selected cases by Masson trichrome and von Kossa stain. Histologically, the type of inflammation was characterized, and the localization and morphology of bacterial colonies were recorded.

Results: A total of 117 cases with chronic thrombosing, valvular endocarditis were received. In 21 cases the lesions extended to the adjacent heart wall (mural endocarditis). Bacteriologically, a dominance of streptococci (obtained in 48.7% (57/117) and dominated by *Streptococcus suis*) and *Erysipelothrix rhusiopathiae* (isolated in 25.6% (30/117)) was cultured. Most lesions were localized in the left side of the hearts, regardless of the aetiology. None of the pigs with *E. rhusiopathiae* infection had lesions in both sides, while this was found in 7% of the streptococci infected pigs. Involvement of more than one valve was seen in 6% of pigs with infection due to *E. rhusiopathiae* and in 27% of pigs with streptococcal infection. Mineralization within lesions was found in 69 cases and was significantly ($P<0.001$) correlated with the presence of granulomatous inflammation (n=27). Typically, a varying number of multinucleated foreign body giant cells was observed adjacent to foci of mineralization and enclosed by concentrically arranged macrophages and epithelioid cells. In lesions caused by streptococci, the inflammatory reaction only contained few neutrophils compared to the lesions with *E. rhusiopathiae*. Characteristically, colonies of streptococci were arranged in distinct groups surrounded by a capsule-like structure, whereas *E. rhusiopathiae* bacteria were present in a more diffuse and cloudy manner. Moreover, the layer of fibrin containing *E. rhusiopathiae* was generally thinner compared to the streptococcal layers.

Discussion: The present study clearly demonstrates that the pathology together with the pattern of colony formation within porcine endocarditis lesions is depending on the aetiology. Moreover, granulomatous endocarditis, which is likely due to a foreign body reaction associated with dystrophic mineralization, has hitherto not been described in cases of porcine endocarditis.

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